

**AMENDMENTS TO THE CLAIMS**

**1-26. (Canceled)**

27. **(Currently Amended)** A method for reducing memory dysfunction associated with damaged hippocampal tissue in a mammal, comprising contacting a hippocampal cell with administering to the mammal a morphogen comprising a conserved C-terminal seven-cysteine skeleton that is one or more of the following:

(a) at least about 60% identical to residues 330-431 of human OP-1 (SEQ ID NO: 2);  
and  
(b) at least about 70% homologous to residues 330-431 of human OP-1 (SEQ ID NO: 2),  
thereby reducing memory dysfunction associated with damaged hippocampal tissue in  
the mammal.

28. **(Previously Presented)** The method of claim 27, wherein said morphogen stimulates synapse formation between hippocampal neurons.

29. **(Previously Presented)** The method of claim 28, wherein said morphogen comprises residues 30-292 of SEQ ID NO:2.

30. **(Previously Presented)** The method of claim 28, wherein said morphogen comprises residues 330-431 of SEQ ID NO:2.

31. **(Previously Presented)** The method of claim 28, wherein said morphogen comprises residues 48-292 of SEQ ID NO:2.

32. **(Previously Presented)** The method of claim 28, wherein said morphogen comprises the amino acid sequence of SEQ ID NO:2.

33. **(Canceled)**
34. **(Currently Amended)** The method of claim 28, wherein said morphogen comprises residues 292 293-431 of SEQ ID NO:2.
35. **(Previously Presented)** The method of claim 28, wherein said morphogen comprises residues 30-431 of SEQ ID NO:2.
36. **(Previously Presented)** The method of claim 28, wherein said morphogen is a BMP-2 polypeptide.
37. **(Previously Presented)** The method of claim 28, wherein said morphogen is a BMP-5 polypeptide.
38. **(Previously Presented)** The method of claim 28, wherein said morphogen is a BMP-6 polypeptide.
39. **(New)** The method of claim 27, wherein the hippocampal tissue damage is caused by a neurotoxin.
40. **(New)** The method of claim 27, wherein the hippocampal tissue damage is caused by transient global ischemia.
41. **(New)** The method of claim 27, wherein the hippocampal tissue damage is caused by permanent global ischemia.
42. **(New)** The method of claim 27, wherein the hippocampal tissue damage is caused by

Traumatic Brain Injury (TBI).

43. (New) The method of claim 27, wherein the morphogen is administered by intraventricular administration.
44. (New) The method of claim 27, wherein the morphogen is disposed in a biocompatible microsphere.
45. (New) The method of claim 27, wherein the damaged hippocampal tissue is damaged from mechanical trauma, chemical trauma, glucose deprivation or a neurotoxin.
46. (New) The method of claim 27, wherein the neurotoxin is selected from ibotenic acid, lead, ethanol, ammonia and formaldehyde.
47. (New) The method of claim 46, wherein the neurotoxin is lead.
48. (New) The method of claim 27, wherein the mammal is afflicted with senility, malnutrition, glucose metabolism disorder, or anorexia.
49. (New) A method for reducing memory dysfunction associated with damaged hippocampal tissue in a mammal, comprising administering to the mammal a morphogen comprising a conserved C-terminal seven-cysteine skeleton that is one or more of the following:
  - (a) at least about 60% identical to residues 330-431 of human OP-1 (SEQ ID NO: 2); and
  - (b) at least about 70% homologous to residues 330-431 of human OP-1 (SEQ ID NO: 2), wherein the hippocampal tissue damage is caused by a neurotoxin, wherein the neurotoxin is lead, wherein the morphogen is disposed in a biocompatible microsphere, thereby reducing memory dysfunction associated with damaged hippocampal tissue in the

mammal.

50. (New) The method of claim 49, wherein the mammal is afflicted with senility.